LETTERS

CIGARETTES AND THE SURGEON GENERAL'S REPORT

"Cigarettes and the US Public Health Service in the 1950s"[1] in the Journal's February issue is not only interesting history. It is also a useful reminder of how outside forces and strongly held individual beliefs can influence what we would like to believe are purely scientific considerations in the promotion of health and the prevention of disease. But I think Dr Parascandola is too dismissive of the importance of the Surgeon General's Report *Smoking and Health*[2] when he notes that the report did not meet some new "evidentiary threshold."

Although it is true that there was little new data, the manner in which the evidence was marshaled, in particular the presentation and elucidation of the 5 criteria for judging the causal significance of an association—that is, the consistency, strength, specificity, temporal relationship, and coherence of the association—made it possible to overcome the resistance of those who insisted—out of honorable or venal motives—that the absence of a blinded prospective trial precluded a judgment of causation.

Despite advances in statistical analysis, the 5 criteria continue to serve as a useful basis for epidemiologic studies where an experimental approach is neither feasible nor ethical. *Smoking and Health* remains excellent reading for introductory courses in epidemiology. ■

Lawrence Bergner, MD, MPH

About the Author

Requests for reprints should be sent to Lawrence Bergner, MD, MPH, 101 W 12th St, 9G, New York, NY 10011 (e-mail: bergner@attglobal.net).

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1. Parascandola M. Cigarettes and the US Public Health Service in the 1950s. *Am J Public Health.* 2001;91:196–205.
2. Advisory Committee to the Surgeon General. *Smoking and Health.* Washington, DC: US Public Health Service; 1964.

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PARASCANDOLA RESPONDS

Dr Bergner's comments are right on target. Although the 1964 surgeon general's report[1] did not present new evidence, it was significant for other reasons. As I noted in my conclusion, Surgeon General Leroy Burney's earlier statements were presented as "opinions" of the Public Health Service.[2] In contrast, under Burney's successor, Luther Terry, the 1964 report was intended to represent the informed judgment of a panel of objective scientists following predetermined rules of inference, and the 5 causal criteria were central to that aim. ■

Mark Parascandola, PhD

About the Author

Requests for reprint should be sent to Mark Parascandola, PhD, Office of Preventive Oncology, Division of Cancer Prevention, National Cancer Institute, Executive Plaza South, Suite T-41, Bethesda, MD 20892 (e-mail: mparasca@erols.com).

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1. Advisory Committee to the Surgeon General. *Smoking and Health.* Washington, DC: US Public Health Service; 1964.
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AIR POLLUTION, WEATHER STRESS, AND BLOOD PRESSURE

Ibald-Mulli et al. recently reported a potentially important association between particulate air pollution and increased blood pressure and stated that this had not been documented previously.[1] In fact, our research group previously reported a positive longitudinal relation between blood pressure and ambient particulate pollution.[2] Our study differed markedly from Ibald-Mulli's in scale, duration, location, atmospheric conditions, and subject characteristics, as shown in Table 1. Nevertheless, certain key results—regression slopes (β) for blood pressure vs particulate pollution concentration—in the 2 studies were similar for our panel with chronic obstructive pulmonary disease (COPD) and for Ibald-Mulli's subgroups with identified risk factors (high plasma viscosity or heart rate). Our

TABLE 1—Effects of Air Pollution on Health: Comparative Results in Ibald-Mulli et al.[1] and Linn et al.[2]

	Ibald-Mulli et al.	Linn et al.
Location	Augsburg, Germany	Los Angeles, US
No. of subjects	2681	30
Age range, y	25 to 64	56 to 83
Health status	Mostly healthy	Severe COPD
Temperature range (°C)	−25 to 19	3 to 35
Pollution range (μg/m^3)	7 to 176 (TSP)	9 to 84 (PM_{10})
Frequency of blood pressure measurements	Twice in 3 years	Daily for 4 days
β [mm Hg/(μg/m^3)]	0.07 to 0.08[a]	0.08 to 0.17

Note. COPD = chronic obstructive pulmonary disease.
[a]In subgroups with identified risk factors.

modestly larger values might reflect our measurement of particles with aerodynamic diameter <10 μm (PM_{10}) rather than total suspended particles (TSP).

As Ibald-Mulli et al. pointed out, weather stresses or gaseous pollutants may confound particle–health relationships. Although we could not study confounders in detail, we found that our subjects' blood pressure appeared to respond to PM_{10} as measured in the outdoor background urban air environment, even though they spent most of their time inside their homes, where particle mass and chemical composition were noticeably different. Again, this seems consistent with the findings of Ibald-Mulli et al., based on outdoor background pollution measurements.

On the basis of this evidence, we recommend wider use of simple noninvasive cardiovascular measurements in studies of air pollution and weather stresses. Increased understanding of response mechanisms, and clearer identification of populations at risk, should result. ■

William S. Linn, MA
Henry Gong Jr, MD

About the Authors

William S. Linn and Henry Gong Jr are with the Environmental Health Service, Rancho Los Amigos National Rehabilitation Center, Downey, Calif, and the Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles. Henry Gong Jr is also with the Department of Medicine, Rancho Los Amigos National Rehabilitation Center, and the Department of Medicine, Keck School of Medicine.

Requests for reprints should be sent to William S. Linn, MA, 51 Medical Science Building, Rancho Los Amigos National Rehabilitation Center, 7601 East Imperial Highway, Downey, CA 90242 (e-mail: linn@hsc.usc.edu).

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1. Ibald-Mulli A, Stieber J, Wichmann HE, Koenig W, Peters A. Effects of air pollution on blood pressure: a population-based approach. *Am. J Public Health.* 2001;91:571–577.

2. Linn WS, Gong H, Clark KW, Anderson KR. Day-to-day particulate exposures and health changes in Los Angeles area residents with severe lung disease. *J Air Waste Manag Assoc.* 1999;49:PM108–PM115.

IBALD-MULLI AND PETERS RESPOND

We welcome the comments from Linn and Gong and are very pleased that they found effects of ambient particulate pollution on blood pressure similar to those found by us and our colleagues.[1] Although the 2 studies differ in their design, they both assessed short-term effects of particulate air pollution on the basis of 24-hour mean pollution concentrations, and we agree that the findings are consistent. Linn and Gong stated that their larger β values might be attributable to the fact that they measured PM_{10} rather than total suspended particles. We think that the absence of control for confounders such as meteorologic variables like temperature and barometric pressure, known to affect blood pressure, should also be considered as a reason for the larger effects found in their study.

We would like to take the opportunity to point out the importance of recent epidemiologic findings that expanded the evidence about the relationship between ambient particulate matter and morbidity, not only in the United States but also in Europe.[2] In particular, recent studies have focused on adverse cardiac outcomes, because previous morbidity and mortality studies showed that acute health risks of particulate matter were associated not only with respiratory causes but also with cardiovascular causes. Based on these findings, several epidemiologic studies are on the way to establishing more consistent evidence of the association between ambient particle exposure and cardiovascular function.

Besides conducting new studies, another approach to gathering evidence of the relationship between ambient particulates and morbidity is to conduct secondary analyses of existing data. Our research group used data collected as part of the World Health Organization's MONICA study (Monitoring Trends and Determinants in Cardiovascular Disease) in Augsburg, Germany, to evaluate the relation between several cardiovascular and blood parameters and air pollution data gathered from existing networks.[1,3,4] Alternatively, data collected on a routine basis could be used to assess the effects of air pollution. In a study conducted by a group at the Harvard School of Public Health, the incidence of cardiac arrhythmias based on data extracted from implanted cardioverter defibrillators[5] was examined in association with air pollution data.

We would like to encourage not only wider use of simple noninvasive cardiovascular measurements to assess the relation of cardiovascular impairment and air pollution but also use of existing data to measure the effects of air pollution on cardiovascular health. ■

Angela Ibald-Mulli, MPH
Annette Peters, PhD

About the Authors

Angela Ibald-Mulli and Annette Peters are with the National Research Center for Environment and Health, Institute of Epidemiology, and the Department of Epidemiology, Ludwig Maximilian University Munich, Neuherberg, Germany.

Requests for reprints should be sent to Angela Ibald-Mulli, GSF–National Research Center for Environment and Health, Institute of Epidemiology, Postfach 1129, 85758 Neuherberg, Germany (e-mail: ibald@gsf.de).

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2. Pope CA III. What do epidemiologic findings tell us about health effects of environmental aerosols? *J Aerosol Med.* 2000;13:335–354.

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4. Peters A, Perz S, Doring A, Stieber J, Koenig W, Wichmann HE. Increases in heart rate during an air pollution episode. *Am J Epidemiol.* 1999;150:1094–1098.

5. Peters A, Liu E, Verrier RL, Schwartz J, et al. Air pollution and incidence of cardiac arrhythmia. *Epidemiology.* 2000;11:11–17.

ERRATUM

In: Sallis JF, Conway TL, Prochaska JJ, McKenzie TL, Marshall SJ, Brown M. The association of school environments with youth physical activity. *Am J Public Health.* 2001;91:618–720.

Incorrect legends appeared with Figures 2 and 3 (p 620). For Figure 2, the legend should read 'High levels of equipment' and 'Low levels of equipment.' For Figure 3, the legend should read 'High levels of improvements' and 'Low levels of improvements.'